# AN OBSERVATIONAL STUDY OF OCULAR MOTOR NERVE PALSIES IN DIABETES MELLITUS

*Dissertation submitted to*
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*In partial fulfillment of the regulations for the award of the degree of*

**M.S. DEGREE BRANCH (III)**
**OPHTHALMOLOGY**

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# CERTIFICATE

This to certify that the dissertation entitled **"AN OBSERVATIONAL STUDY OF OCULAR MOTOR NERVE PALSIES IN DIABETES MELLITUS"** is a bonafide original work of **Dr. R. SARAVANAN,** in partial fulfillment of the requirements for **M.S. Degree Branch – III (OPHTHALMOLOGY)** Examination of the Tamilnadu Dr.M.G.R Medical University to be held in March 2009.

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# ABBREVIATIONS

| | | |
|---|---|---|
| AGE | - | Advanced glycation end products |
| BP | - | Blood pressure |
| CT | - | Computerised axial tomography |
| DAG | - | Diacylglycerol |
| DM | - | Diabetes mellitus |
| EOM | - | Extra ocular movements |
| FBS | - | Fasting blood sugar |
| HbA1c / GHb | - | Glycosylated haemoglobin A1c |
| LPS | - | Levator palpebrae superioris |
| MRA | - | Magnetic resonance angiography |
| MRI | - | Magnetic resonance imaging |
| NAD | - | Nicotinamide adenine dinucleotide |
| NADP | - | Nicotinamide adenine dinucleotide phosphate |
| NADPH | - | Reduced nicotinamide adenine dinucleotide phosphate |

| | | |
|---|---|---|
| PAI - 1 | - | Plasminogen activator inhibitor - 1 |
| PKC | - | Protein kinase C |
| PPBS | - | Post prandial blood sugar |
| RAGEs | - | Receptors for advanced glycation end products |
| ROS | - | Reactive oxygen species |
| TGF $\beta_1$ | - | Transforming growth factor $\beta_1$ |
| VDRL | - | Venereal disease research laboratory |
| VEGF | - | Vascular endothelium derived growth factor |

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## PART – I



## PART II



## PART III

# INTRODUCTION & HISTORY

A perfect alignment between the motor system of two eyes is responsible for viewing an object as single. The extraocular muscles of both eyes work in co-ordination. When any one or more of these falter, it may manifest as double vision, drooping of eyelids, deviation of eyes or some times with pain. Patients may present to the ophthalmologist for one of these complaints, may be referred by another physician or be seen accidently while they come for a routine check up. This may be one of the first manifestation of a multisystem disease like diabetes mellitus.

The best early evidence of description of diabetes in world's literature is recorded in *Ebers papyres* dated 1550 BC. Arateus of Cappadocia coined the term *'diabetes'*, meaning, *"siphon"*, to explain the liquefication of flesh and bone into urine.

In 400 B.C. *Susrata*, an Indian Surgeon had described the diabetic syndrome as characterised by a *"honeyed urine"*.

Diabetic retinopathy was first described in 1869 by Eduard von Jauger, and specific lesions like – "Microaneurysms and new vessels" – were described by Stephan Mackenzie and Edward Nettleship in 1880.

In 1964, Marchal de calvi associated neuropathy with diabetes and symptoms were clearly reported by Frederide Pavy in 1885. Albuminuria was noted as a common abnormality in diabetic patients by Joslin in 1916.

# DEFINITION

Diabetes mellitus is defined as a group of metabolic diseases characterized by hyperglycaemia resulting from defects in insulin secretion, insulin action or both. The chronic hyperglycaemia is associated with long-term damage, dysfunction and failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels.

# CLASSIFICATION OF DIABETES MELLITUS

**Type 1** (β-cell destruction usually leading to absolute insulin deficiency)

Autoimmune

Idiopathic

**Type 2**

Ranges from predominantly insulin - resistant with relative insulin deficiency to a predominantly insulin - secretory defect with or without insulin resistance.

**Other specific types**

Genetic defects β - cell function

Genetic defects of insulin action

Diseases of exocrine pancreas

Endocrinopathies

Drug-induced or chemical - induced

Infections

Uncommon forms of immune mediated diabetes

Other genetic syndromes sometimes associated with diabetes

Gestational diabetes

# BIOCHEMICAL MECHANISMS OF DIABETES TISSUE DAMAGE

- Chronic tissue damage in diabetes is generally related to the severity and duration of hyperglycemia. Other determinants of specific complications include genetic predisposition and hypertension. Tissue damage may continue to evolve even after hyperglycaemia has been improved (hyperglycaemic memory).

- Diabetes particularly affects tissues in which glucose uptake increases during hyperglycaemia, leading to raised intracellular glucose concentrations. High glucose levels may cause cumulative and progressive tissue damage through irreversible alterations of structural proteins and other long-lived molecules, or (e.g. in the retina) through the summation of micro vascular occlusions.

- At cellular level, hyperglycaemia may damage tissues by enhanced glucose flux through the polyol pathway; formtion of advanced glycation end-products (AGEs); activation of protein kinase (PKC); and stimulation of the hexosamine pathway. All these mechanisms may stem ultimately from

overproduction of superoxide by mitochondria, which metabolize excess glucose.

- The polyol pathway, whose rate-limiting enzyme is aldose reductase, reduces glucose and several other sugars (e.g. glucose to sorbitol). The pathway operates in tissues that express aldose reductase (e.g. lens, retina, endothelium), particularly during hyperglycaemia. Cellular damage may result from enhanced production of glycating sugars (e.g. methylglyoxal) and thus AGE formation, and/or depletion of reduced glutathione (a scavenger of reactive oxygen species, ROSs), resulting in oxidative damage.

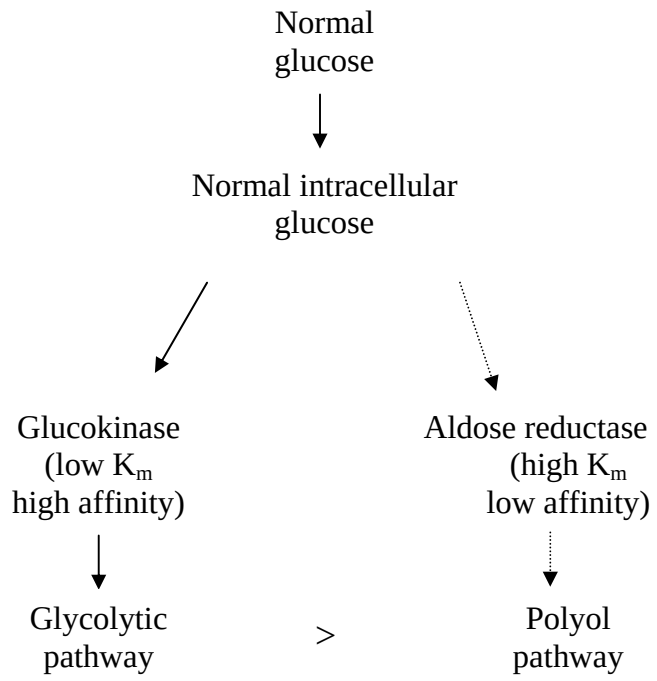
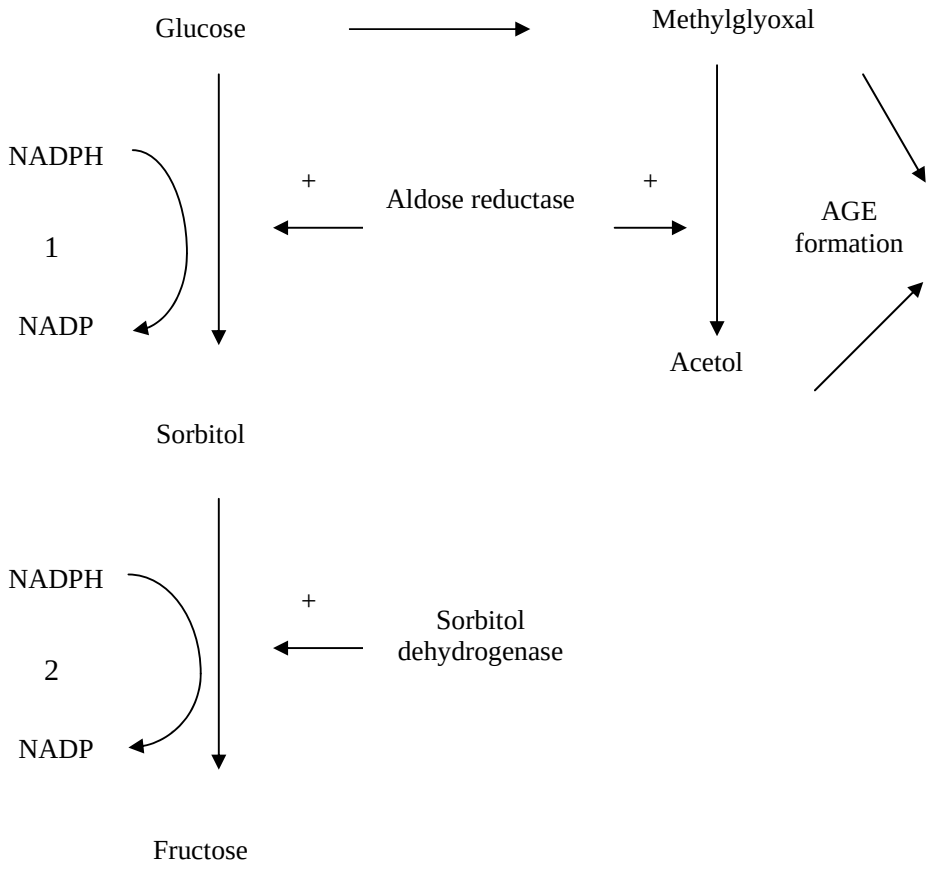



**Fig 1 a: The Polyol pathway**

The pathway is normally inactive, but becomes active when intracellular glucose levels rise.




1. ↓ NADPH causes
   - Reduced glutathione depletion
   - Oxidative damage
2. ↑ NADH / $NAD^+$ causes
   - Increase Triose phosphates (AGE formation)
   - PKC activation

**Fig. 1 b: Polyol Pathway**

Consequences of increased glucose flux through polyol pathway include the generation of powerful glycating sugars (methylglyoxal, acetol and triose phosphates), enhanced oxidative damage and protein kinase C (PKC) activation.

- AGEs are the irreversible products of proteins that react with glycating sugars such as 3 - deoxy glucosone, methylglyoxal and the relatively weak glucose. Covalent cross-linking and other changes damage structural proteins (e.g. collagen) and extracellular matrix components, while circulating AGE - modified proteins bind to specific receptors for advanced glycation endproducts (RAGEs) on macrophages and endothelial cells. Macrophages release proinflammatory cytokines, while endothelial cells express procoagulant and adhesion proteins that favour thrombosis and ultimately atheroma formation; endothelial expression of vascular endothelium-derived growth factor (VEGF) also increases vascular permeability.

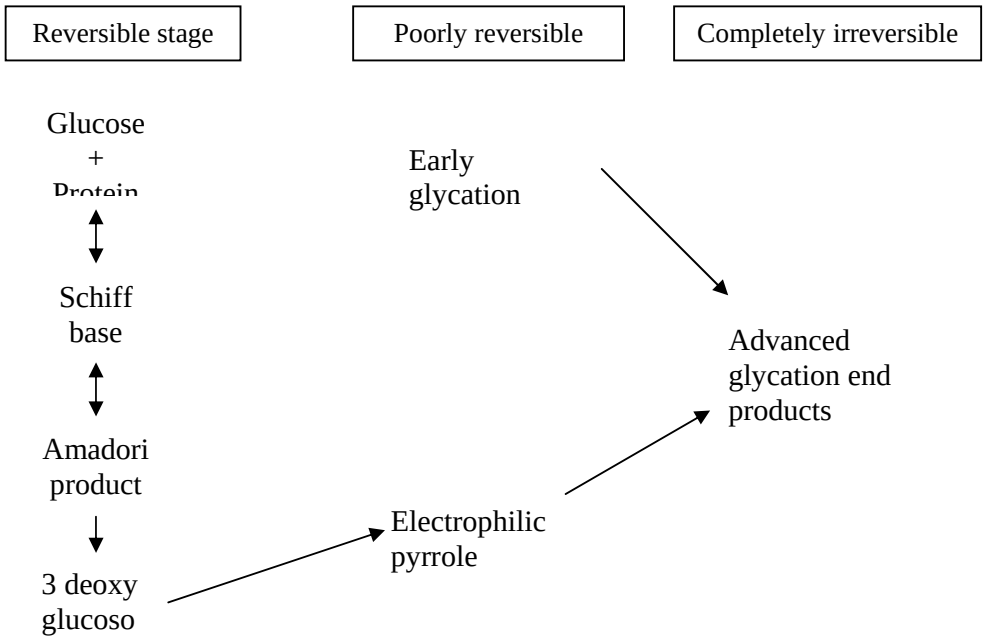



**Fig. 2:**

Formation of reversible, early**,** non-enzymatic glycation products and of irreversible advanced glycation end products (AGEs). Through a complex series of chemical reactions, Amadori products can form families of imidazole based and pyrrole - based glucose derived cross - links.

PKC insoforms (especially β and δ) are activated by diacyl glycerol (DAG), synthesized de-novo from increased intracellular glucose. This may decrease tissue blood flow (by inhibiting production of the potent vasodilator, nitric oxide) and enhance vascular permeability via increased VEGF expression. Induction of plasminogen activator inhibitor - 1 (PAI - 1) expression may favour thrombosis.

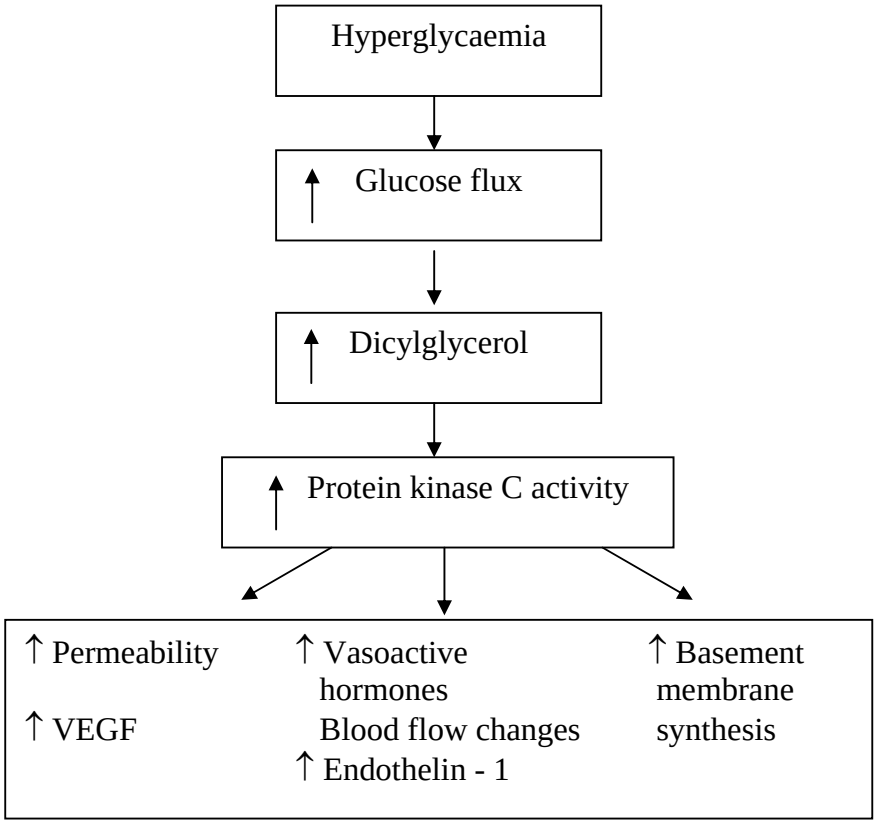



**Fig. 3**

Activation of protein kinase - C by de-novo synthesis of diacylglycerol, following increased glucose utilization.

- High intracellular glucose levels result in increased production of glucosamine, which by leading to glycation of transcription factors (forming their O - Glc Nacylated derivatives) may enhance transcription of specific genes including PAI - 1 and transforming growth factor $\beta_1$ (TGF-$\beta_1$).

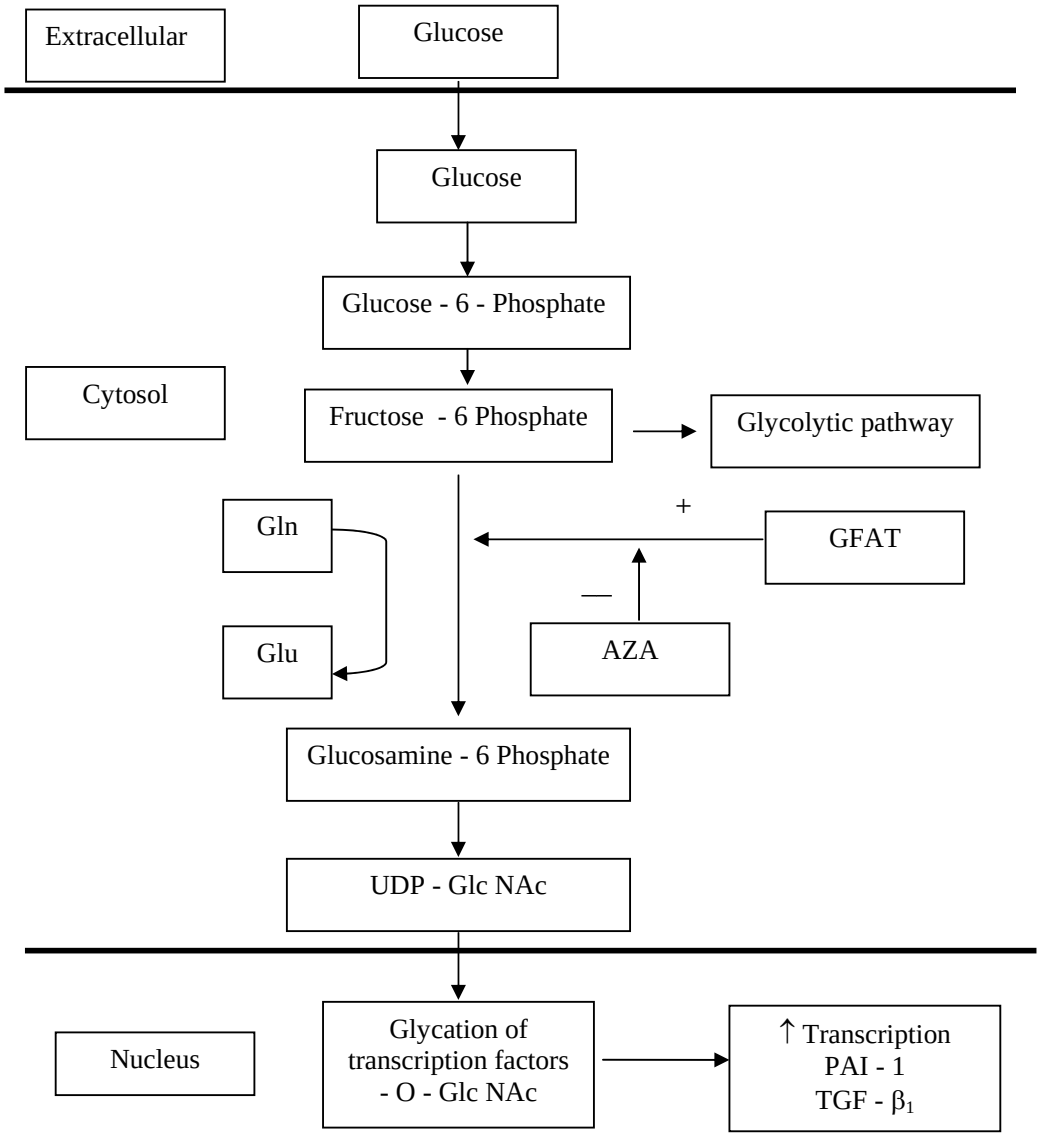



**Fig. 4 The Glucosamine pathway**

Glucosamine - 6 phosphate, generated from fructose - 6 phosphate and glutamine (Gln), is converted to UDP - N acetyl glucosamine, which can glycate transcription factors and thus enhance transcription of genes including PAI - 1 and TGF $\beta_1$ Glutamine: fructose - 6 phosphate amidotransferase (GFAT), the rate - limiting enzyme is inhibited by azaserine (AZA).

- Excess mitochondrial production of the ROS, superoxide, may cause all the above abnormalities. Superoxide production via the tricarboxylic acid (TCA) cycle is increased by hyperglycaemia; consequences include stimulation of aldose reductase activity, enhanced formation of methylglyoxal and thus AGE, increased DAG synthesis and PKC activation, and hexosamine pathway overactivity.

## OCULAR MANIFESTATIONS OF DIABETES MELLITUS

| S.No. | STRUCTURE | MANIFESTATIONS |
|---|---|---|
| **EXTRAOCULAR** | | |
| 1. | LIDS | Ptosis, Xanthelasma chronic blepharitis Recurrent stye & chalazion |
| 2. | EXTRAOCULAR MUSCLES | Mononeuropathy |
| 3. | LACRIMAL APPARATUS | Decreased tear secretion, increased tear glucose levels |
| 4. | ORBIT | Orbitorhinomucormycosis |
| **OCULAR** | | |
| 1. | CORNEA | Corneal hypoaesthesia, Recurrent erosions, corneal abrasions, punctate keratopathy, neurotrophic keratitis |
| 2. | IRIS AND PUPIL | Rubeosis iridis, small pupil, neovascular glaucoma |
| 3. | ANGLE STRUCTURES | Open angle glaucoma, secondary glaucoma |
| 4. | LENS | Fluctuating myopia, snowflake cataract, senile cataract |
| 5. | VITREOUS | Posterior vitreous detachment, Asteroid bodies |
| 6. | RETINA | Diabetic Retinopathy, Lipaemia Retinalis, Decreased contrast sensitivity and colour vision |
| 7. | OPTIC NERVE | Anterior ischaemic optic neuropathy, optic atrophy |

These are the associated findings in diabetes. We can look for them, so that they aid in clinical diagnosis of ocular motor nerve palsies.

# OCULOMOTOR NERVE

## ANATOMY

The third cranial nerve is entirely motor in function. It supplies all the extraocular muscles of the eyeball except the lateral rectus and superior oblique. It also supplies the intraocular muscles namely the sphincter pupillae and the ciliary muscles.

## FUNCTIONAL COMPONENTS

1. SOMATIC EFFERENT - concerned with movements of the eyeball.

2. GENERAL VISCERAL EFFERENT (parasympathetic) - for accommodation and contraction of the pupil.

3. GENERAL SOMATIC AFFERENT - for carrying proprioceptive impulses from the muscles supplied by the third nerve.

## THE OCULOMOTOR NUCLEAR COMPLEX

### LOCATION

It is situated in the midbrain at the level of the superior colliculus in the ventromedial part of the central gray matter that surrounds the cerebral aqueduct.

It is a longitudinal column, 10 mm long extending above from the floor of the third ventricle and below it is related to the nucleus of the fourth nerve. There are two motor nuclei.

1. Main motor nucleus of large multipolar neurons
2. Accessory Edinger Westphal nucleus of small multipolar neurons.

**The main motor nucleus has the following subnuclei:**

1. DORSOLATERAL NUCLEUS - supplies ipsilateral inferior rectus
2. INTERMEDIATE NUCLEUS - supplies ipsilateral inferior oblique

3. VENTROMEDIAL NUCLEUS - supplies ipsilateral medial rectus

4. PARAMEDIAL NUCLEUS - supplies contralateral superior rectus

5. CAUDAL CENTRAL NUCLEUS - supplies bilateral levator palpebrae superioris.

The Edinger Westphal nucleus lies posterior to the main oculomotor nuclear mass. It consists of a median and two lateral parts. It gives rise to preganglionic parasympathetic fibres.

**CONNECTIONS OF THE NUCLEUS**

1. Cerebral cortex

   - motor cortex of both sides through the corticonuclear tracts.

   - Visual cortex through the superior colliculus.

   - frontal eye field

2. Nuclei of $4^{th}$, $6^{th}$ and $8^{th}$ cranial nerves through the medial longitudinal fasciculus.

3. Pretectal nucleus of both sides

4. Vertical and torsional gaze centres

5. Cerebellum through the vestibular nuclei.

**COURSE AND DISTRIBUTION**

It can be divided into four parts

1. The fascicular part

2. The basilar part

3. The intracavernous part

4. The intraorbital part

**THE FASCICULAR PART**

It consist of efferent fibres that pass from the third nerve nucleus through the red nucleus and the medial and a small lateral root, which unite to from a flattened nerve, which then gets twisted bringing the inferior fibres superiorly and vice versa. Thus the nerve becomes a rounded cord. The nerve then passes between the posterior cerebral and superior cerebellar arteries. Then it runs forward in the interpreduncular cistern (running lateral and parallel to the posterior communicating artery) to reach the cavernous sinus.

## THE INTRACAVERNOUS PART

The nerve enters the cavernous sinus by piercing the posterior part of its roof on the lateral side of the posterior clinoid process. It then descends on the lateral wall of the sinus, where it lies above the trochlear nerve. In the anterior part of the cavernous sinus, the nerve divides into superior and inferior divisions which enter the orbit through the middle part of the superior orbital fissure within the annulus of Zinn.

## THE INTRAORBITAL PART

In the orbit the smaller superior division ascends on the lateral side of the optic nerve and supplies the superior rectus and levator palpebrae superioris. The larger inferior division divides into three branches.

i. Nerve to medial rectus passes inferior to the optic nerve.

ii. Nerve to inferior oblique (longest of the three branches) passes in between the inferior rectus and lateral rectus and supplies the oblique from its posterior border. It gives off the motor root to the ciliary ganglion.

iii. Nerve to inferior rectus passes and enters the muscle on its upper aspect.

**THE FEATURES OF THIRD NERVE PALSY**

It may be complete or incomplete and it may be congenital or acquired.

1. Ptosis - due to paralysis of LPS.

2. Deviation - eyeball is turned down, out and slightly intorted due to unopposed action of the lateral rectus and the superior oblique.

3. Ocular movements - restriction of the following movements.

i. Adduction - due to paralysis of medial rectus.

ii. Elevation - due to paralysis of superior rectus and inferior oblique.

iii. Depression - due to paralysis of inferior rectus

iv. Extortion - due to paralysis of inferior rectus and inferior oblique.

4. Pupil - is fixed and dilated due to paralysis of sphincter pupillae.

5. Accommodation - completely lost due to paralysis of ciliary muscle.

6. Crossed diplopia - appears on manually raising the eyelid, which occurs due to paralytic divergent squint.

7. Head posture - if the pupillary area is uncovered the head takes a posture consistent with the directions of actions of paralysed muscle i.e. head is turned to the opposite side, titled towards the same side and chin is slightly raised.

**PUPIL SPARING ISOLATED III NERVE PARESIS**

The pupillomotor fibres of the III nerve travel in the outer layers of the nerve and are therefore closer to the nutrient blood supply enveloping the nerve. The outer fibres are supplied by the pial plexus whereas the inner fibres are supplied by the vasa nervosum.

So this explains why the diabetics (where the vasa nervonum are affected) have pupillary sparing in 80% and similarly in any ischaemic vascular etiology. In contradiction when compressive lesions involve the III nerve the superficial fibres are affected resulting in pupillary involvement in 90%.

Most patients with ischaemic III nerve paresis demonstrate improvement in motility measurements within one month or may have complete recovery by 3 months (maximum : 6 months).

Cranial imaging like MR scanning – MRI, MRA, Four vessel angiography and Lumbar puncture are recommended if:

i. The pupil is involved i.e. dilates or becomes dilated in the initial 5 – 7 days after onset.

ii. No significant improvement in 3 months.

iii. The patient develops signs of aberrant regeneration of III nerve.

iv. Other neurologic findings develop.

**ABERRANT REGENERATION OF III NERVE**

This is seen after trauma and tumour compression of the III nerve, but never after an ischaemic III nerve paresis. If the patient is followed with a presumed diagnosis of ischaemic III nerve palsy and then develops signs of aberrant regeneration, then MR scanning and cerebral angiography are indicated.

# TROCHLEAR NERVE

The trochlear nerve is entirely motor in function and supplies only the superior oblique muscle of the eyeball.

## PECULIARITIES

- The only cranial nerve to arise from the dorsal aspect of the brain.
- The only cranial nerve to cross completely to the other side i.e. the trochlear nerve arises from the contralateral nucleus.
- The longest and thinnest of all cranial nerves.

## FUNCTIONAL COMPONENTS

1. SOMATIC EFFERENT – concerned with the primary, secondary and tertiary actions of superior oblique.
2. GENERAL SOMATIC AFFERENT – carries proprioceptive impulses from the superior oblique. The impulses are relayed to the mesencephalic nucleus of the trigeminal nerve.

## NUCLEUS

Situated in the ventromedial part of the central gray matter of the midbrain at the level of inferior colliculus. It is continuous with the III nerve nuclear complex. It belongs to the somatic efferent column of nuclei.

## CONNECTIONS OF THE NUCLEUS

1. Cerebral cortex
    i. Motor cortex – of both sides through the corticonuclear tracts.
    ii. Visual cortex – through the superior colliculus
    iii. Frontal eye fields.
2. Nuclei of 3rd, 6th and 8th cranial nerves through the medial longitudinal bundle.
3. Superior colliculi through the descending predorsal bundle.
4. Vertical and torsional gaze centres.
5. Cerebellum through the vestibular nuclei.

## COURSE AND DISTRIBUTION

It is divided into

i. The fascicular part

ii. The precavernous part

iii. The intracavernous part

iv. The intraorbital part

## THE FASCICULAR PART

It consists of efferent fibres which after leaving the nucleus, pass posteriorly around the aqueduct in the central gray matter and decussate completely in the anterior medullary velum.

## THE PRECAVERNOUS PART

The trochlear nerve trunk emerges from the superior medullary velum just below the inferior colliculus on the dorsal aspect of midbrain. It then winds round the superior cerebellar peduncle and the cerebral peduncle just above the pons. It runs beneath the free edge of the tentorium, and like the III nerve passes between the posterior cerebral and superior cerebellar arteries to appear ventrally lateral to cerebral

peduncle. It then pierces the dura on the posterior corner of the roof of the cavernous sinus to enter into it.

**THE INTRACAVERNOUS PART**

In the cavernous sinus, the nerve runs forwards in its lateral wall lying below the III nerve and above the first division of the fifth cranial nerve. In the anterior part of the cavernous sinus, it rises, crosses over the III nerve and leaves the sinus to pass through the lateral part of the superior orbital fissure (where it passes superolateral to the annulus of Zinn and medial to the frontal nerve).

**THE INTRAORBITAL PART**

After entering through the lateral part of the superior orbital fissure, the nerve passes medially above the origin of the LPS and ends by supplying the superior oblique on its orbital surface.

The number of fibres in the intraorbital part of the trochlear nerve are greater than its intracranial part. These extra fibres carrying the proprioceptive impulses from the superior oblique leave the trochlear nerve to join the ophthalmic division of fifth nerve in the cavernous sinus.

## FEATURES OF IV NERVE PALSY

1. Hyperdeviation due to weakness of superior oblique. This becomes more obvious when the head is titled towards ipsilateral shoulder (Park Bielchowsky head tilt test).

2. Ocular movements – depression is limited in adduction. Intorsion is also limited.

3. Diplopia – vertical diplopia occurs on looking down.

4. Abnormal head posture – To avoid diplopia head adopts a posture such that the action of superior oblique is less needed i.e. face is slightly turned to opposite side, chin is depressed and head is titled towards the opposite side.

## PARK-BIELCHOWSKY'S THREE STEP TEST

The medial and lateral rectus muscles do not have a vertical action. Therefore hypertropia of paretic etiology is due to weakness of one or more of the following vertically acting muscles. If the hypertropia is due to weakness of only one of these eight muscles, answering the following three questions identifies the paretic muscle.

1. First step – which is the higher eye?

   a) If the patient has a right hypertropia then the weak muscle is either a depressor of the right eye (right inferior rectus / right superior oblique) or an elevator of the left eye (left superior rectus / left inferior oblique).

   b) If the patient has left hypertropia then the weak muscle is either an elevator of the right eye (right superior rectus / right inferior oblique) or depressor of the left eye (left inferior rectus are left superior oblique).

2. Second step – hypertropia worse on right or left gaze?

   - The vertical rectus muscles (superior and inferior recti) have their greatest vertical action (and least torsional action) when the eye is abducted. The oblique muscles (superior and inferior obliques) have their greatest vertical action (and least torsional action) when the eye is adducted.

   So in each case.

   i. Right hypertropia worse on right gaze (right inferior rectus / left inferior oblique).

ii. Right hypertropia on left gaze (right superior oblique / left superior rectus).

iii. Left hypertropia worse on right gaze (left superior oblique / right superior rectus).

iv. Left hypertropia worse on left gaze (right inferior oblique / left inferior rectus).

3. Third step – Is the hypertropia worse on head tilt to right or left?

a. The superior muscles (superior rectus and superior oblique) intort the eye; the inferior muscles (inferior rectus and inferior oblique) extort the eye.

b. When the head is titled to the right, right eye will be intorted by the contraction of the right superior rectus and right superior oblique; these two muscles work together in effecting the intorsion and neutralize each other's vertical action (right superior rectus is an elevator and right superior oblique is a depressor).

c. If one of these muscles is the paretic muscle responsible for the hypertropia, then the vertical action will not be neutralized and the hypertropia will be worse on tilting the head to the right shoulder.

# ABDUCENT NERVE

It is an entirely motor nerve that supplies the lateral rectus muscles of the eyeball.

## FUNCTIONAL COMPONENTS

i. SOMATIC EFFERENT – for lateral movement of the eye.

ii. GENERAL SOMATIC AFFERENT for proprioceptive impulses from the lateral rectus muscle. These impulses ultimately reach the mesencephalic nucleus of the trigeminal nerve.

## NUCLEUS

Situated in the lower part of pons, close to the midline beneath the floor of the IV ventricle. It is closely related to the fasciculus of the facial nerve. It consists of two types of multipolar cells-large and small. The large multipolar cells give rise to fibres of the abducent nerve, while the fibres of the small multipolar cells relay in the oculomotor nucleus via the medial longitudinal fasciculus. The small multipolar cells are believed to form the paraabducent nucleus. Since the abducent nucleus belongs to the group of somatic efferent nuclei, it lies in line with the nuclei of IV and III nerves above and the hypoglossal nucleus below.

## CONNECTIONS OF THE NUCLEUS

1. Cerebral cortex

    i. Motor cortex (precentral gyrus) through the afferent corticonuclear fibres from both cerebral hemispheres.

    ii. Visual cortex, through the superior colliculus.

    iii. Frontal eye fields

2. Nuclei of III, IV and VIII cranial nerves through the medial longitudinal bundle.

3. Pretectal nucleus of both sides.

4. Horizontal gaze centre through the medial longitudinal bundle.

5. Cerebellum through vestibular nuclei.

## COURSE AND DISTRIBUTION

It is divided into

i. The fascicular part

ii. The basilar part

iii. The intracavernous part and

iv. The intraorbital part.

**THE FASCICULAR PART**

It consists of efferent fibres which start from the nucleus, pass forward traversing the medial lemniscus and the pyramidal tract. These then emerge by 7-8 rootlets from the junction of pons and medulla just lateral to the pyramidal prominence of medulla. The rootlets join to form one nerve, at varying distances from the origin.

**THE BASILAR PART**

The nerve then runs forwards, upwards and slightly laterally through the cisterna pontis between the pons and the clivus. The nerve then runs upwards on the back of petrous temporal bone near its apex. At the sharp upper border of the petrous bone, the nerve bends forward at right angles under the petrosphenoidal ligament through the Dorello's canal and enters the cavernous sinus by piercing its posterior wall at a point lateral to the dorsum sellae and superior to the apex of petrous temporal bone.

## THE INTRACAVERNOUS PART

In the cavernous sinus, the nerve runs horizontally forward, occupying a position below and lateral to the internal carotid artery. The internal carotid artery is surrounded by the sympathetic plexus. The nerve then leaves the cavernous sinus to enter the orbit through the middle part of the superior orbital fissure through the annulus of Zinn. In the superior orbital fissure, the abducent nerve lies inferolateral to the oculomotor and nasociliary nerves.

## THE INTRAORBITAL PART

In the orbit the nerve runs forwards and enters the ocular surface of the lateral rectus muscle just behind its middle portion after dividing into three or four branches.

## CLINICAL FEATURES OF SIXTH NERVE PALSY

1. Deviation – In the primary position, the eyeball in convergent due to unopposed action of the medial rectus muscle.

2. Ocular movements – Abduction is restricted.

3. Diplopia – Uncrossed horizontal diplopia occurs, worse towards the action of the paralysed muscle.

4. Head posture – The face is turned towards the action of the paralysed muscle to minimize diplopia.

# FEATURES OF OCULAR MOTOR NERVE PALSIES IN DIABETES

- III nerve commonly affected
- More common in elderly
- Pupillary sparing

Because the peripherally situated pupillary fibres supplied by the pial plexus are spared whereas the centrally located fibres supplied by vasa nervonum are affected.

- Usually recover spontaneously and completely in months.
- Can manifest as multiple episodes of transient ophthalmoplegia affecting different muscles of either one or both eyes.
- Ocular motor nerve palsies in diabetes can be painless or painful

## DIFFERENTIAL DIAGNOSIS OF PAIN LESS OPHTHALMOPLEGIA

Diabetes

Hypertension

Atherosclerosis

Weber's syndrome

Tumours of orbit

**DIFFERENTIAL DIAGNOSIS OF PAINFUL OPHTHALMOPLEGIA**

Cavernous sinus thrombosis

Tolosa Hunt syndrome

Mucormyosis

Nasopharyngeal carcinoma

Herpes Zoster

Lymphoma

# TREATMENT OF OCULAR MOTOR NERVE PALSIES

Management of Diabetes mellitus mainly consists of

1. Life style modification

2. Tight glycemic control with insulin

Follow up of cases of ocular motor nerve palsy that do not need urgent management, like the posterior communicating artery aneurysm must be at 6 weekly intervals till 6 months or till two consecutive 6 weeks follow-ups reveal no change in motility. Every time diplopia charting, Hess charting, recording of deviations in nine gazes is done. During the meantime, patient is greatly disturbed by diplopia. So some nonsurgical modalities are practiced for symptomatic relief. It no resolution occurs after about 8-12 months then surgery is considered.

1. Prisms - are helpful in providing binocular vision as well as reducing the changes of development of contracture, but are useful only in small angle squints. Fresnel prisms are also used.

2. Botulinum toxin – the ipsilateral antagonist is paralysed by chemodenervation. The effect lasts for about 2-3 months. If necessary the injection can be repeated.

3. Occlusive prisms or opaque contact lens.

4. Surgery – mainly to weaken the antagonist, usually ipsilateral and sometimes also the contralateral antagonist, in addition to strengthening the paralysed muscle. The amount of recession resection varies depending upon which eye habitually fixates (secondary deviation or primary deviation needs to be corrected). Another principle is to restrain the contralateral antagonist by performing retroequatorial myopexy.

In the case of III nerve, the aim is to achieve diplopia free ocular position in primary position and downgaze. The latter should never be compromised for the upgaze. Anyway it is difficult because the III nerve supplies most of the extraocular muscles except two. Moreover aberrant regenerations alter the clinical picture. Each case has to be considered on an individual basis.

In the case of IV nerve, either strengthening of superior oblique or weakening of ipsilateral inferior oblique or contralateral inferior rectus

is done. The results of surgery for both congenital and acquired IV nerve palsy is excellent.

# AIM OF THE STUDY

1. To study the ocular motor nerve palsy pattern due to Diabetes Mellitus

2. To study the relationship of glycaemic control in ocular motor nerve palsies.

3. To study the association of diabetic retinopathy and nephropathy in case of ocular motor nerve palsies.

4. To study the recovery pattern.

# MATERIALS AND METHODS

The cases studied, included those patients with neurogenic ocular motor nerve palsies who presented to the Regional Institute of Ophthalmology and Govt. Ophthalmic Hospital. All age groups and both sexes were included.

A complete ophthalmological workup was done.

## INCLUSION CRITERIA

1. All infranuclear ocular motor nerve palsies with DM

## EXCLUSION CRITERIA

All supranuclear, nuclear nerve palsies, myogenic and restrictive neuropathies. Associated combined condition like heart disease, were excluded.

## REGISTRATION

Name

Age

Sex

Occupation

Address

## HISTORY OF PRESENT ILLNESS

The common complaints were:

a. Double vision-whether uniocular/binocular, constant/ intermittent, fluctuating or not, more for near or distance, whether images were horizontally or vertically separated, whether it is increased on any particular direction, onset and progress.

b. Pain-headache/preiorbital pain, location, nature, any radiation, aggravating and relieving factors, any association with nausea/vomiting.

c. Drooping of lids – unilateral/bilateral, total / partial

d. Defective vision – apart from double vision, any blurring or inability to see due to drooping of lid.

e. Deviation of eyeball-right/left, eye, duration

f. Abnormal head posture

g. Vertigo (sensation of rotation of self/surroundings)

Details of the progress from onset, the treatment undergone to the present state is noted. Any other significant medical/surgical history is also recorded.

**PAST HISTORY**

H/o diabetes, hypertension, tuberculosis, syphilis, AIDS, malignancy in the present or past.

H/o migraine or neurologic disease

H/o xanthems and vaccination

**PERSONAL HISTORY**

Diabetes, smoking, alcoholism etc.

**GENERAL EXAMINATION**

General vital data like pulse, blood pressure, peripheral pulses are noted. Also gives an idea of the health status of the patients.

## OCULAR EXAMINATION

- Head posture, facial symmetry are noted.
- Any deviation of eyeball is recorded. Under slit lamp, details of the anterior segment from the lids to the lens are noted.
- Extraocular movements are noted down-both ductions and versions. While looking for EOM, the aberrant innervation patterns are also looked for.
- Pupil size, reaction, any anisocoria is noted.
- A dilated fundus examination and refraction is done. Ptosis and proptosis if present are evaluated.
- Diplopia charting – is done in a dark room. Patient is asked to wear goggles with red in front of the right eye and green before the left eye. A torch light with a stenopaeic slit is used. The patient is asked to look at the torch held 120 cm away and then the torch is moved to various positions. The patient is asked to describe the position of the images. The false image is usually the fainter and farther one. Any tilt of the image and variation in the distance between images at various position is asked for.

- If a superior oblique palsy is suspected, Parks Bielchowsky's 3 step head tilt test is done.

- A forced duction test is performed in doubtful cases to rule out restrictive etiology.

- Tensilon test is performed in some cases to rule out myasthenia gravis.

**NEUROLOGIC EXAMINATION**

Examination of other cranial nerves

Motor, sensory, cerebellar symptoms and signs.

**EXAMINATION OF THYROID**

Any neck swelling is looked for

**EXAMINATION OF SPINE & BACK**

To look for congenital anomalies and neurocutanous markers.

**EXAMINATION OF ENT STRUCTURE**

**INVESTIGATIONS**

Both right and left eye (for all cases)

1. Vision
   a. Uncorrected (Using Snellen's charts at 6 metres)
   b. Best corrected

2. Intraocular pressure–with applanation tonometer after topical anaesthesia

3. Detailed slit lamp examination

   Lid

   Conjunctiva

   Cornea

   Iris

   Pupil

   Anterior Chamber

   Lens/Pseudophakia/Aphakia

4. Fundus examination-any abnormalities, diabetic retinopathy etc.

5. Diplopia charting

6. Park 3 step test

7. Measurement of deviation-primary & secondary deviation, cover uncover test in various gaze positions, for near and distance as well.

8. Hess charting

9. Exophthalmometry

10. Visual field examination

**BLOOD TEST** : (For all cases)

Total count

Differential count

Erythrocyte sedimentation rate

Blood sugar – Fasting

Postprandial

In doubtful cases, Glucose tolerance test/Hb A1c

Mantoux intradermal test

Serum cholesterol

Blood VDRL

Rheumatoid factor

**RADIOLOGY** (in indicated cases)

X ray orbit – fractures/erosions

X ray skull

X ray orbit – fractures/erosion

X ray skull

X ray chest – tuberculosis

X ray PNS – (paranasal sinuses) – mucococle, antral growth, sinusitis, orbit floor fractures.

ORBITAL USG – (in indicated cases)

NEURO IMAGING: (in indicated cases)

CT

MRI

MRA

Cerebral angiography

Specialist opinion (in indicated cases)

Diabetologist

Otorhinolaryngologist

Neurophysician/Neurosurgeon

Radiologist

**FOLLOW UP**

Recording of patient's complaints-whether stable/improving worsening.

- Vision
- Pupil assessment
- Extraocular movements

- Diplopia charting

- Fundus

- Examining for signs of pupillary involvement or aberrant regeneration in cases of III N palsy

- Investigations

  Blood sugar FBS

  PPBS

  Hb A1c

  BP

  Imaging studies, if necessary.

# RESULTS

51 cases of diabetic ocular motor nerve palsies were examined. A prospective study was conducted.

## 1. AGE DISTRIBUTION

The following table shows the age distribution in the various ocular motor nerves affected due to diabetes.

**TABLE – I**

| Age group | III Nerve | IV Nerve | VI Nerve | Multiple Nerves | Total |
|---|---|---|---|---|---|
| 21-30 | 0 | 0 | 1 | 0 | 1 |
| 31-40 | 1 | 0 | 3 | 0 | 4 |
| 41-50 | 1 | 0 | 7 | 1 | 9 |
| 51-60 | 12 | 0 | 17 | 0 | 29 |
| 61-70 | 4 | 0 | 4 | 0 | 8 |
| | | | | | **51** |

In the study of 51 patients, the patients affected with sixth nerve palsy were maximal (62.74%) followed by III nerve palsy (35.29%) and the least in frequency were multiple ocular motor nerve palsy (1.96%). There was were no patient with fourth nerve involvement.

Regarding the age distribution, considering all the nerve palsies in total, the maximum number of patients belonged to 51-60 years age group (56.86%) followed in frequency by 41-50 years age group with 17.6% of patients, 61-70 years age group with 15.68% patients and 31-40 years age group with 7.84% patients. The least number was seen in the age group of 20-30 years (1.96%).

Considering each nerve palsy, with regard to third nerve, the maximum number was in the age group of 51-60 years (23.52%) and followed in frequency by the age group of 61-70 years (7.84%).

With regard to sixth nerve palsy the maximum number was in the age group of 51-60 years. (33.33%) followed by 41-50 years age group (13.7%).

Multiple cranial ocular motor nerves were affected in one patients of the age group 41-50 years.

## 2. SEX DISTRIBUTION

**TABLE - II**

| Nerve | Male | Female | Total |
|---|---|---|---|
| III | 7 | 11 | 18 |
| IV | 21 | 11 | 32 |
| Multiple | 1 | 0 | 1 |
| Total | 29 | 22 | 51 |

In the study there was a slight gender difference, with males out numbering females – 56.8% males against 43.13% females. The incidence of III nerve palsy was higher in the females (21.56%) compared to males (13.7%). With regard to VI nerve palsy, the incidence was higher among males (41.17%) compared to female (21.56%). The patient with multiple nerve palsy was a male patients. The ratio of III nerve to VI nerve involvement among males was 1:3 where as in female it was 1:1.

## 3. LATERALITY

**TABLE - III**

| Nerve | Right | Left |
|---|---|---|
| III | 7 | 11 |
| VI | 13 | 19 |
| Multiple | 0 | 1 |
| Total | 20 | 31 |

Left eye involvement was common, with 31 out of 51 patients presenting with left side nerve palsy (60.70%). 19 patients of sixth nerve palsy and 11 patient with third nerve palsy were left sided. Multiple cranial nerve palsy patients was affected in the left side. Laterality difference was not statistically significant.

## 4. GLYCAEMIC CONTROL AND MICROVASCULAR COMPLICATIONS IN III NERVE PALSY

**TABLE - IV**

| HbA1c% | III Nerve | Urine – Alb | Retinopathy |
|---|---|---|---|
| <6 | 1 | 0 | 1 |
| 6.1 – 8.0 | 8 | 0 | 2 |
| 8.1 – 10.0 | 8 | 2 | 5 |
| > 10 | 1 | 1 | 1 |

In this study at the time of presentation of the patient to our hospital the glycaemic control was assessed by glycosylated haemaglobin levels. The table shows the incidence of nerve palsy along with the microvascular complications as assessed by urine macro albuminuria for diabetic nephropathy and the retinopathy status assessed by fundus examination. The glycosylated haemaglobin level gives the glycaemic status of the patient during the last three months.

It was observed that, with regard to III nerve palsy with $HbA_{1C}$ less than 6, one patient developed retinopathy. In the group of 8 patients with $HbA_{1C}$ 6.1-8.0 two had retinopathy (25%). With HbA1c between 8.1-10, 5 of 8 patients had retinopathy (62.5%) where as 2 had nephropathy (25%) Only one patient had $HbA_{IC}$ > 10 and had both nephropathy and retinopathy (100%).

## 5. GLYCAEMIC CONTROL AND MICROVASCULAR COMPLICATIONS IN VI NERVE PALSY

**TABLE - V**

| HbA1c% | VI Nerve | Urine – Alb | Retinopathy |
|---|---|---|---|
| <6 | 1 | 0 | 0 |
| 6.1 – 8.0 | 19 | 3 | 6 |
| 8.1 – 10.0 | 7 | 0 | 3 |
| > 10 | 1 | 1 | 1 |

With regard to VI nerve palsy, patients with $HbA_{IC}$ less than six showed no nephropathy or retinopathy. In the group of 19 patients with $HbA_{IC}$ between 6.1-8.0, six patient had retinopathy (31.57%), and 3 patients had nephropathy (15.78%).

7 patients had $HbA_{IC}$ between 8.1-10.0 and of these 3 had retinopathy (42.85%) and none had nephropathy. Only one patient presented with $HbA_{IC}$ of more than 10.0%, and had nephropathy (100%) and retinopathy (100%) both.

One patient with multiple palsy i.e. III & VI nerve involvement had HbA1C of 9% and showed presence of both retinopathy and nephropathy.

## 6. RECOVERY PATTERN

**TABLE - VI**

| Recovery | III | VI | Multiple | Total |
|---|---|---|---|---|
| Full | 10 | 20 | 0 | 30 |
| Partial | 3 | 7 | 1 | 11 |
| No Recovery | 1 | 2 | 0 | 3 |
| Lost follow up | 4 | 3 | 0 | 7 |

The recovery pattern of the ocular motor nerve palsy due to Diabetes. Mellitus was varied. The recovery was noted when the patients came for follow up. Follow up was done at 2-3 weekly intervals for 3-4 months.

Few cases (ie. 7 patients) were lost to follow up during their reference to diabetology department and department of neuromedicine. So the recovery could not be documented in these patients. The recovery was noted in 4 months in most cases, which was almost complete in 6 months.

# DISCUSSION

## 1. AGE

In this study 51 cases of Diabetes mellitus with ocular motor nerve palsies were examined. The widest range was associated with sixth nerve palsies in the present series. The majority of patients with either III nerve (23.52%) or VI nerve palsy (33.33%) belonged to 51-60 years age group. In a study of 22 cases of III nerve palsy by Jack. E Goldstein and David G. Cogan the average age was 62 years. The sixth nerve was the commonly affected nerve in this study. Whereas, in literature the third nerve palsy is the most commonly affected followed by the sixth and seventh nerve. And in the third nerve palsy the pupil is usually spared. Cause is thought to be vascular with a localized infarct involving the brain stem nuclei or the emerging nerve root. Older people are mainly affected.

## 2. SEX

In this study there was an overall slight gender difference with male preponderance 56.86% males compared to 43.13% females. Incidence of third nerve palsy was observed to be higher in females (21.56%) compared to males (13.7%) whereas incidence of sixth nerve

palsy was more among males (41.17%) compared to females (21.56%). The patient with multiple nerve palsy was a male patient. The ratio of III nerve to sixth nerve involvement among males was 1:3. Whereas in females it was 1:1.

In our series the male to female ratio for incidence of III nerve palsy was 1:1.57 and for sixth nerve palsy it was 1.9:1. Comparing with the study by Goldstein and Cogan for third nerve palsy where male to female ratio was 1:1.

## 3. LATERALITY

In the present series left eye involvement was found to be common (60.78%) 37.25% patients had sixth nerve palsy, 21.56% patients had third nerve palsy and 1.96% was multiple cranial nerve palsy. Bilaterality was not found in our series. The laterality does not seem to have a significance comparing with the study of Goldstein & Cogan where right & left eye were equally affected. There was a left eye preponderance in our study.

## 4. GLYCAEMIC CONTROL AND MICROVASCULAR COMPLICATIONS IN III NERVE PALSY

Glycosylated Haemoglobin was taken to assess the overall glycaemic control of the patient at the time of incidence of the specific cranial nerve palsy. There is a large body of evidence that Glycated Haemoglobin relates to integrated preceding glycemic control. It is now recognised that Glycated Haemoglobin is the weighted measure of preceding glyceamia with recent events contributing more than distant ones.

Because of the link established by the DCCT and UKPDS between control as measured by HbA1c and microvascular complications, the magnitude of the HbA1c percentage can be regarded as a risk factor for the development of microangiopathy.

The American Diabetes Association recommends the goal of diabetes therapy should be an HbA1c of < 7%.

In our study only one patient was seen with third nerve palsy with HbA1c of less than 6%. This implies overall good glycaemic control in the past three months but this patient had mild non proliferative diabetic

retinopathy which may be explained by the duration of diabetes being more than 12 years.

There were 8 patients with HbA1c between 6.1% to 8.0% of whom 25% had retinopathy and none had nephropathy. In the group with HbA1c between 8.1 to 10.%, there were 8 patients, of whom 62.5% had retinopathy and 25% had nephropathy.

Only one patient in this series had HbA1c of more than 10% and she had both nephropathy and retinopathy. This implies that the patients with poorer control indicated by higher HbA1c had higher insidence of microvascular complications like nephropathy and retinopathy but there was no correlation between HbA1c and ocular motor cranial nerve palsies.

## 5. GLYCAEMIC CONTROL AND MICROVASCULAR COMPLICATIONS IN VI NERVE PALSY

With regard to VI nerve only one patient had HbA1c less than 6% and had no evidence of retinopathy or nephropathy. This patient had diabetes for 2 years.

In the group with HbA1c between 6.1% to 8.0% there were 19 patients of whom retinopathy was found in 6 patients (31.8%) and 3 patients had nephropathy (15.78%).

When glycated haemoglobin was in the range of 8.1 to 10%, 7 patients had VI nerve palsy with 3 patients having retinopathy (42.85%) and none had nephropathy. The only patient with HbA1c of more than 10% had both nephropathy and retinopathy. The sole patient with multiple palsy had HbA1c level of 9% with both microvascular complication (i.e. retinopathy and nephropathy) This again went in for favour to imply that glyceamic control did not relate to oculor motor cranial nerve palsies.

## 6. RECOVERY PATTERN

30 patients (58.8%) showed full recovery of whom 10 had III nerve palsy (55.5%) and 20 had VI nerve palsy (62.5%).

Partial recovery in the series was seen in 21.5% patients of whom 3 suffered with III nerve palsy (16.6%) and 7 with VI nerve palsy (21.87%).

No recovery was seen in 5.88% of total patients of whom one belonged to III nerve palsy (5.55%) and 2 with VI nerve palsy (6.25%).

7 patients were lost to follow up. Patients who showed no recovery by the end of 6 months were further evaluated. In the series recovery pattern was found to be good with more than three fourth of the patients recovering fully or partially.

# CONCLUSION

1. The diabetic ocular motor nerve palsies occur in a wide range of age but are more common in the age group of 51 - 60 years.

2. Overall males were affected more than females except in third nerve palsy which showed a slight female preponderance.

3. Left eye involvement was common but was of no significance.

4. Sixth nerve palsy was more common than third nerve palsy.

5. Fourth nerve involvement in diabetes mellitus is comparatively rare & no patients were detected with IV nerve palsy.

6. There was no significant correlation between the level of glycaemic control and incidence of ocular motor nerve palsy though retinopathy and nephropathy were seen to occur more with poorer glycaemic status.

7. More than three fourth of the patients had complete or partial recovery.

8. A careful history, general and complete ophthalmological workup with necessary basic investigations is enough to diagnose patients with diabetic ocular motor nerve palsies. Further evaluation and specialist opinion are necessary when there is deviation from the normal pattern of recovery.

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# PROFORMA

Name :

Age :

Sex :

Occupation :

Address :

Date : Clinic No. Hospital No.

Complaints :

History:

1. Visual complaints
2. Diplopia, Headache
3. Nausea
4. Vomiting
5. Consciousness
6. Gait
7. Sphincteric control
8. Weakness
9. Injury
10. Personality disturbances

**Examination**

Visual Acuity :

| | **Right Eye** | | | | | **Left Eye** | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | VA UA | Sph | Cyl | Axis | VA | VA UA | Sph | Cyl | Axis | VA |
| DV | | | | | | | | | | |
| NV | | | | | | | | | | |

Head position : Head tilt / Face turn / Chin / Other skull abnormalities

Eyelids : Ptosis, Retraction, Lidlag, Marcus Gunn Phenomenon

Eye Position : Deviation / Proptosis

Movements : Uniocular & binocular, conjugate or disconjugate

Nystagmus :

| | **Right Eye** | **Left Eye** |
|---|---|---|
| Pupils: | | |
| Fundus: | | |
| Tension: | | |
| Fields: | | |

**Central Nervous System**

**Higher Functions:**

| | Right Eye | Left Eye |
|---|---|---|
| Crainal Nerves:<br><br>(1,2,5,7,8,9,10,11,12 & 3,4,6) | | |
| Motor System: Tone, Power, Wasting, Involuntary movements | | |
| Sensory system: Touch, Pain, Temperature, Position & vibration | | |
| Cerebellar System: Co-ordination, Tandem Walking | | |
| Reflexes : | | |

Hess Screening, Diplopia Charting (Separate Charts Attached)

Lab Investigations

Blood Routine

TC

DC

ESR

X - Ray No & Date

Blood Sugar fasting and post prandial

Urine Routine

Mantoux

VDRL

Special Investigations : (CT, MRI)

Treatment:

Follow up:

# KEY TO MASTER CHART

LE: Left Eye

RE: Right Eye

NAD: No Abnormal Diagnosis

Mild NPDR: Mild Non Proliferative Diabetic Retinopathy

Mod NPDR: Moderate Non Proliferative Diabetic Retinopathy

HTR: Hypertensive Retinopathy

POST PRP: Post Pan Retinal Photocoagulation

N: Negative

P: Positive

FBS: Fasting Blood Sugar

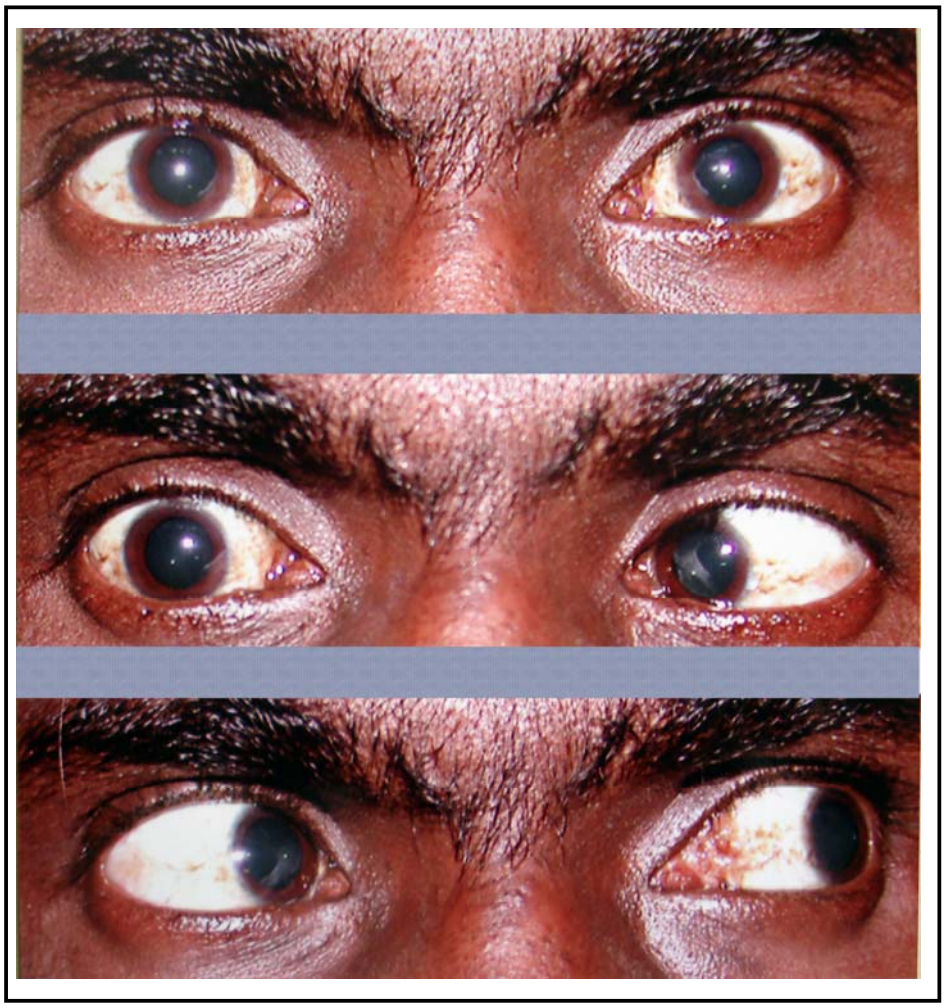
PPBS: Postprandial Blood Sugar

Hb1Ac: Glycated haemoglobin

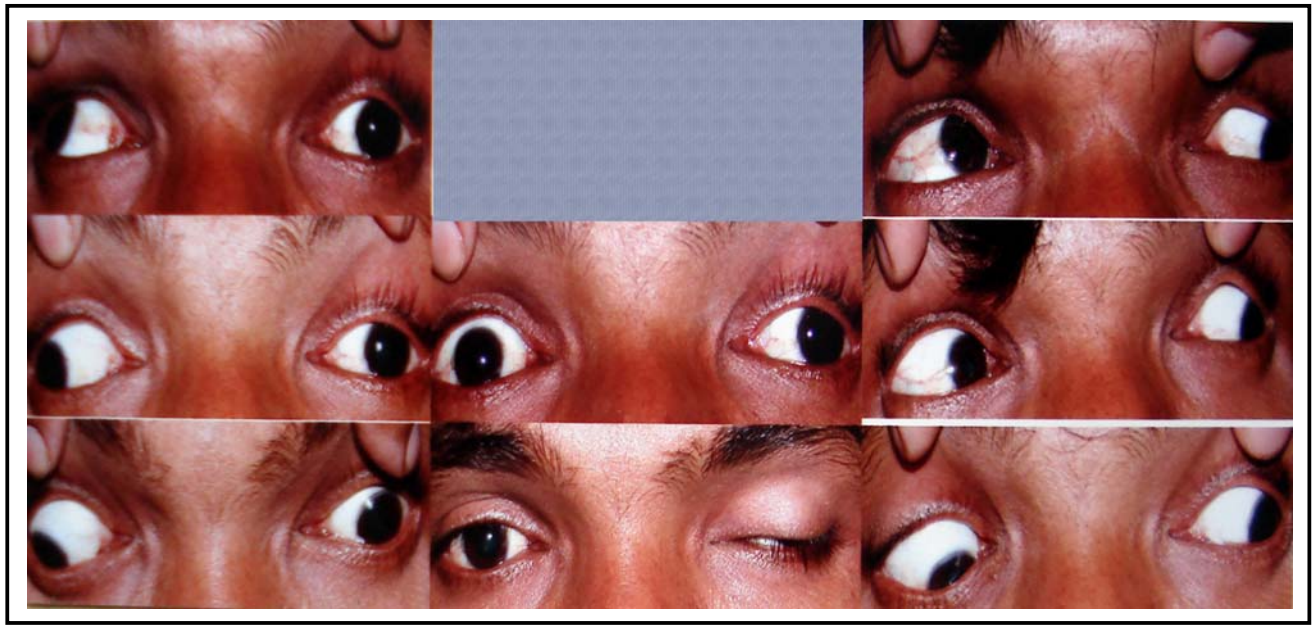
## LIST OF SURGERIES PERFORMED

| S. No | NAME | AGE/ SEX | IP NO | DIAGNOSIS | PROCEDURE |
|---|---|---|---|---|---|
| 1. | Sivaprakasam | 65/M | 382619 | RE – IMC | RE-SICS/PCIOL |
| 2. | Leelavathy | 67/F | 382759 | BE–IMC L>R | RE-SICS/PCIOL |
| 3. | Sivagamy | 70/F | 382947 | BE–IMC L>R | LE-SICS/PCIOL |
| 4. | Rubavathy | 60/F | 390756 | BE–IMC R>L | RE-SICS/PCIOL |
| 5. | Gopal | 42/M | 391787 | BE-IMC R>L | RE-SICS/PCIOL |
| 6. | Krishnan | 70/M | 393782 | LE- PHACOLYTIC GL | RE-SICS/PCIOL |
| 7. | Savithri | 58/F | 390999 | BE–IMC R>L | RE-SICS/PCIOL |
| 8. | Selvaraj | 59/M | 396179 | LE –IMC | LE-SICS/PCIOL |
| 9. | Elumalai | 65/M | 399979 | RE –IMC LR-PCIOL | RE-SICS/PCIOL |
| 10. | Narayanan | 70/M | 392147 | LE –IMC | LE–SICS/PCIOL |
| 11. | Velusamy | 52/M | 392436 | BE–IMC L>R | RE–SICS/PCIOL |
| 12. | Syed Nazar | 65/M | 399423 | BE-IMC L>R | LE–SICS/PCIOL |
| 13. | Gowri | 52/F | 399415 | LE -MC RE-PCIOL | LE–SICS/PCIOL |
| 14. | John | 68/M | 399442 | BE-IMC R>L | RE–SICS/PCIOL |
| 15. | Ayisha Bee | 55/F | 399961 | BE-IMC R>L | RE–SICS/PCIOL |
| 16. | Devi | 65/F | 400200 | RE –IMC | RE–PHACO/PCIOL |
| 17. | Ananthraman | 58/M | 400474 | RE –IMC | RE–PHACO/PCIOL |
| 18. | Indrani | 52/F | 400576 | RE –IMC | RE–PHACO/PCIOL |
| 19. | Muniammal | 60/F | 394493 | RE-FUNGAL CORNEAL ULCER WITH HYPOPYON | RE-TKP 9MM OVER 8MM |
| 20. | Ravi | 17/M | 25789 | LE CHALAZION | LE – I & C |
| 21. | Kaveri | 37/F | 27240 | RE-PTERYGIUM | EXCISION |
| 22. | Rajammal | 35/F | 384392 | LE-FUNGAL CORNEAL ULCER WITH HYPOPYON | LE – AC WASH WITH AMP – B |
| 23. | Muniappan | 59/M | 83120 | LE-PAN OPHTHALMITIS | LE-EVISCERATION |
| 24. | Shanthi | 52/F | 376292 | RE-ABSOLUTE GLAUCOMA | RE-CYCLOCRYO |
| 25. | Lakshmi | 50/F | 396830 | RE-CHR. DACROCYSTITIS | RE DCR |
| 26. | Babu | 09/M | 396857 | RE-TRAUMATIC ENDOPHTHALMITIS | RE-INTRA VITREAL INJECTION OF BROAD SPECTRUM ANTBIOTICS |
| 27. | Malakondiah | 55/M | 387906 | BE-SEC ACG | RECOMBINED SUR |
| 28. | Girija | 45/F | 402574 | BE-PACG | RE-AG SURGERY |
| 29. | Shankar | 32/M | 402574 | RE-CHR. DACROCYSTITIS | RE – DCR |
| 30. | Jaibunisa | 15/F | 397265 | RE-OLD RHEG.RD | RE-RD SURG (360º ENCIRCLAGE WITH CRYO WITH SRF DRAINAGE) |

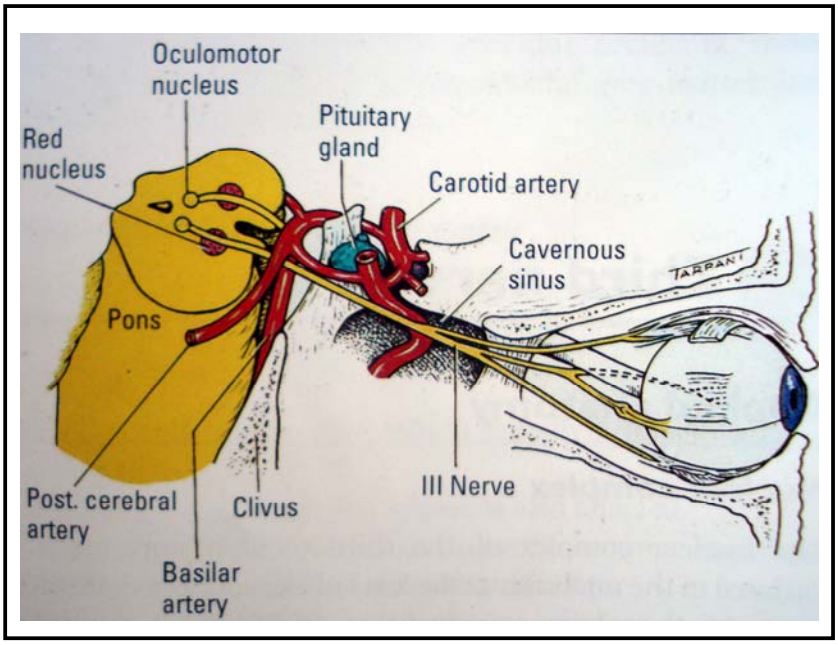
**RIGHT SIXTH NERVE PALSY**



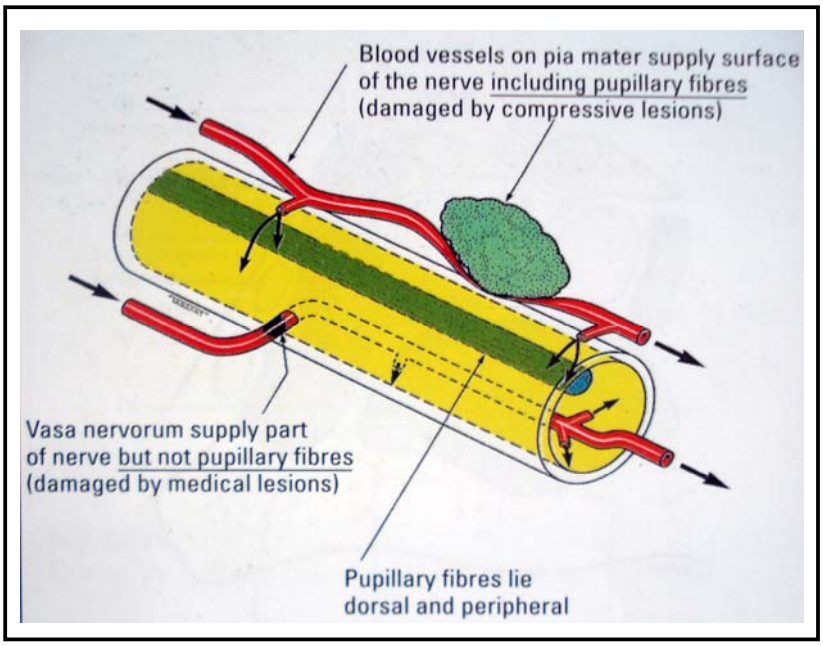
## LEFT OCULOMOTOR NERVE PALSY



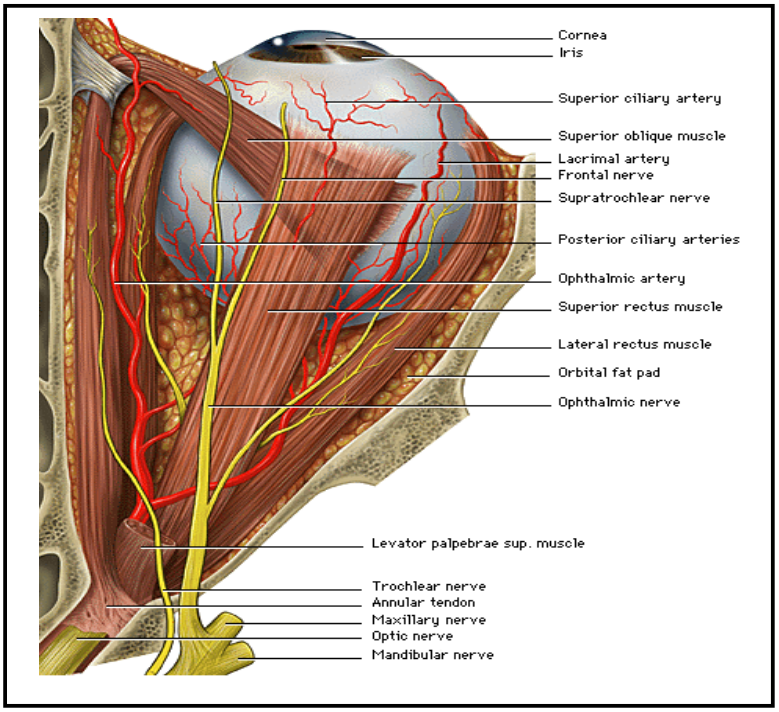
## LATERAL VIEW OF THE COURSE OF THIRD NERVE



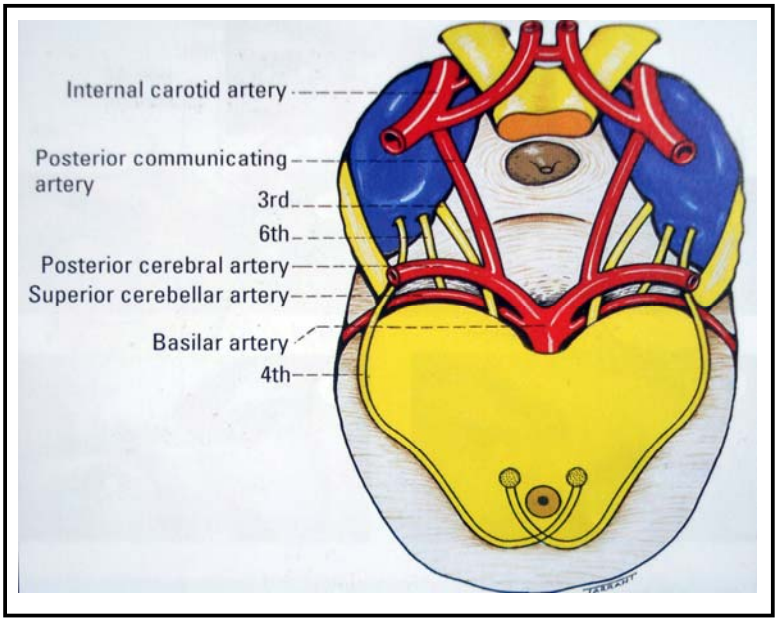

## LOCATION OF PUPILLOMOTOR FIBRES WITHIN `THE TRUNK OF THE THIRD NERVE



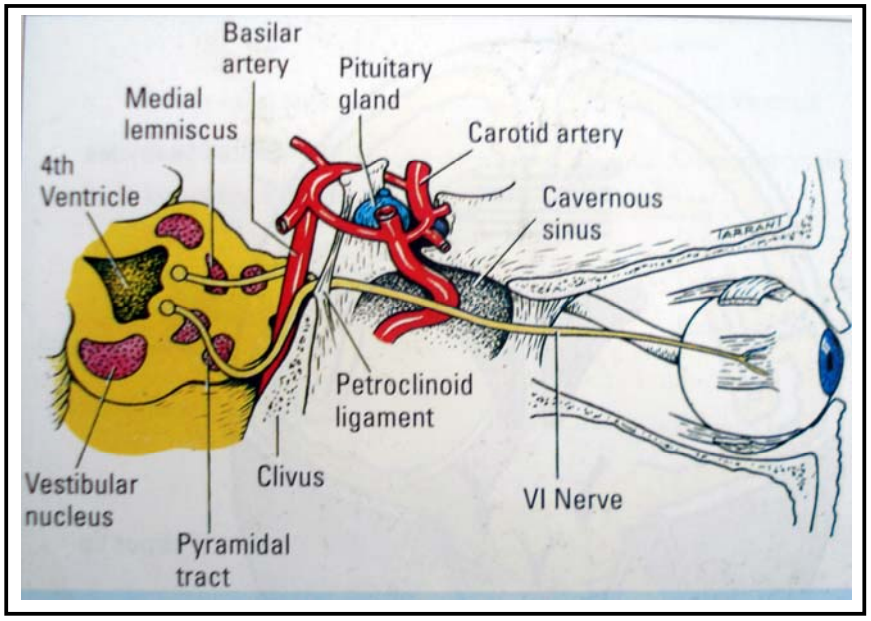
# TROCHLEAR NERVE



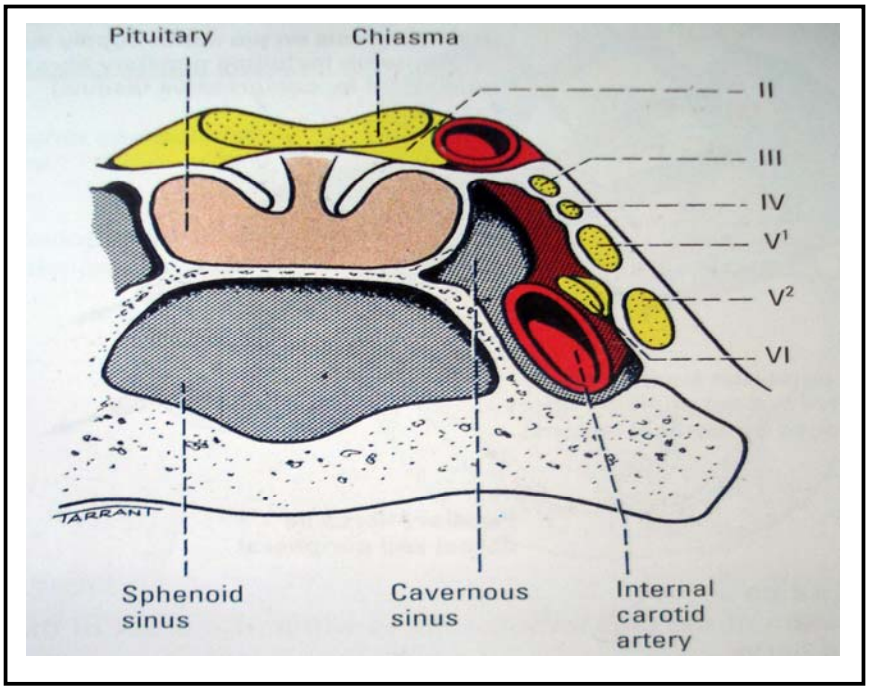

# DORSAL VIEW OF THE COURSE OF TROCHLEAR NERVE



## LATERAL VIEW OF THE COURSE OF SIXTH NERVE




## LOCATION OF THE CRANIAL NERVES IN THE CAVERNOUS SINUS VIEWED FROM BEHIND

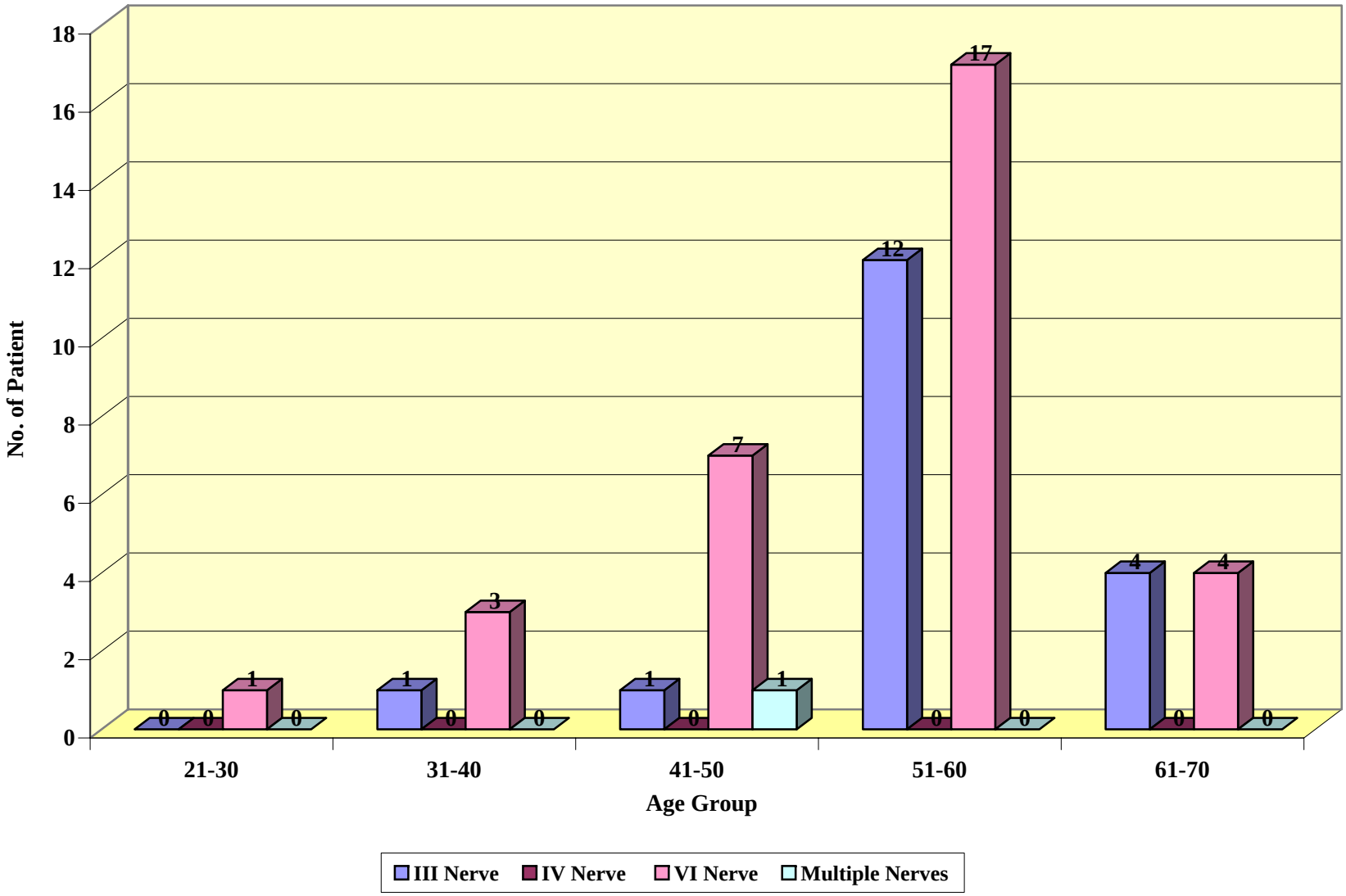


## MASTER CHART

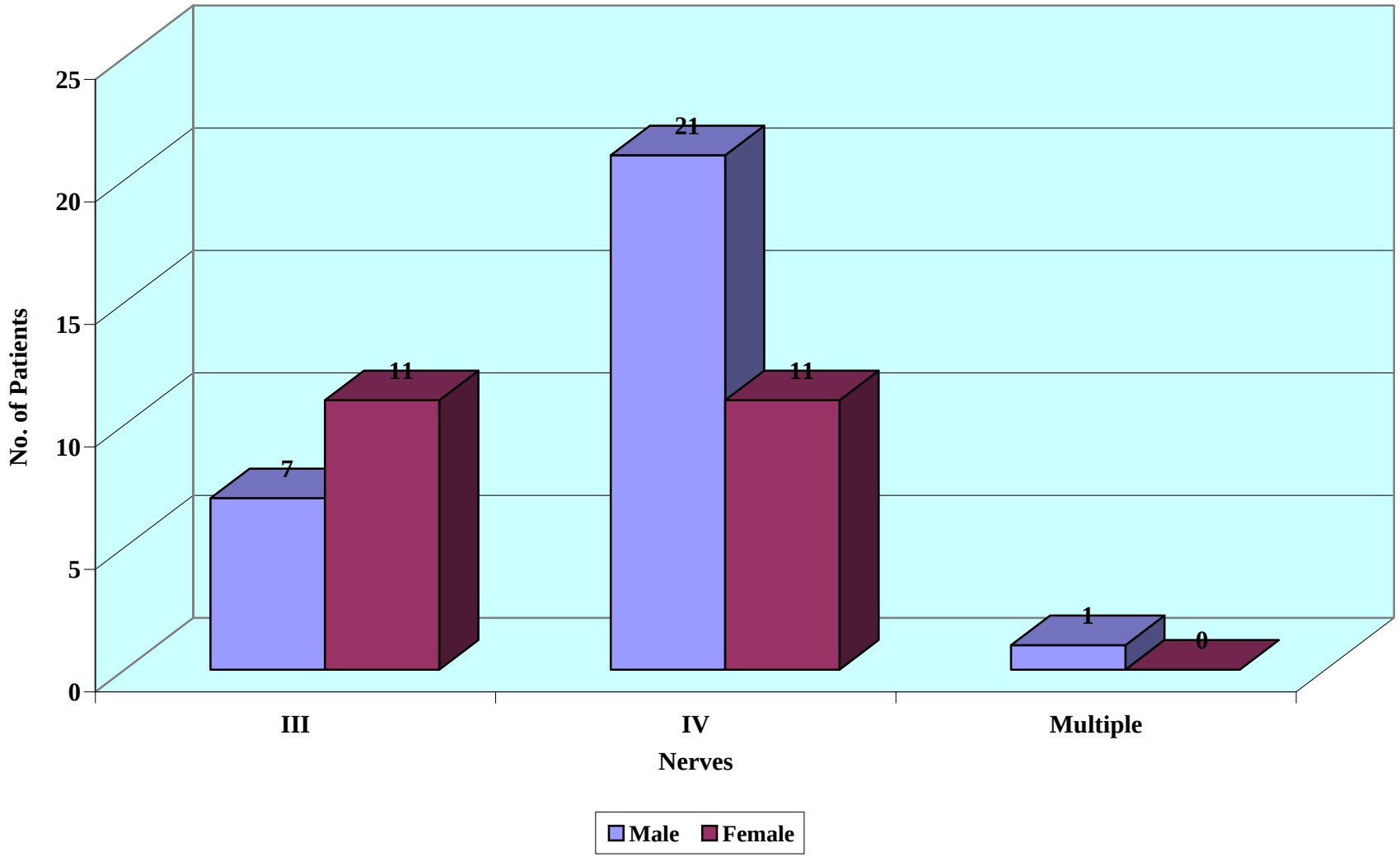
| S.No | Name | Age | Sex | Laterality | Complaints | FBS | PPBS | Fundus | III N | VI N | Urine Alb | Recovery | HbA1c |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Durai | 55 | M | LE | Diplopia | 128 | 165 | NAD | N | P | N | Full | 6.5 |
| 2 | Rajalakshmi | 48 | F | LE | Diplopia | 152 | 212 | HTR | N | P | N | Full | 7.5 |
| 3 | Ramachandran | 54 | M | LE | Diplopia | 100 | 180 | NAD | N | P | N | Full | 7 |
| 4 | Krishnan | 50 | M | RE | Diplopia | 132 | 198 | NAD | N | P | N | Lost | 8 |
| 5 | Jayamma | 60 | F | LE | Drooping | 180 | 270 | ModNPDR | P | N | P | Full | 10.5 |
| 6 | Rani | 55 | F | LE | Drooping | 112 | 224 | NAD | P | N | N | Partial | 9.5 |
| 7 | Aftab | 50 | M | LE | Drooping | 145 | 202 | ModNPDR | P | P | P | Partial | 9 |
| 8 | Chinnaiya | 53 | M | LE | Diplopia | 130 | 165 | POST PRP | N | P | P | Full | 7.5 |
| 9 | Indrani | 60 | F | RE | Diplopia | 98 | 137 | NAD | N | P | N | Full | 6.5 |
| 10 | Naduman bee | 45 | F | LE | Diplopia | 136 | 168 | NAD | N | P | N | Full | 6.7 |
| 11 | Chandrasekar | 70 | M | RE | Diplopia | 111 | 214 | NAD | N | P | N | Lost | 6 |
| 12 | Pandurangan | 66 | M | LE | Diplopia | 144 | 200 | Mild NPDR | N | P | P | Partial | 7.5 |
| 13 | Meenakshi sundaram | 58 | M | RE | Diplopia | 106 | 152 | NAD | N | P | N | Full | 5.8 |
| 14 | Vanaja | 54 | F | LE | Diplopia | 178 | 252 | ModNPDR | N | P | N | Partial | 9 |
| 15 | Christie raj | 56 | M | LE | Drooping | 125 | 186 | Mild NPDR | P | N | N | Lost | 8.5 |
| 16 | Kuppammal | 44 | F | RE | Drooping | 212 | 380 | ModNPDR | P | N | P | Full | 10 |
| 17 | Ester | 53 | F | RE | Drooping | 102 | 156 | NAD | P | N | N | Full | 6.2 |
| 18 | Sundarajan | 48 | M | LE | Diplopia | 143 | 198 | ModNPDR | N | P | N | Full | 7 |
| 19 | Natarajan | 52 | M | RE | Diplopia | 322 | 486 | Mild NPDR | N | P | P | No recovery | 11 |
| 20 | Sokkanayaki | 65 | F | RE | Drooping | 80 | 136 | Mild NPDR | P | N | N | Lost | 5.5 |
| 21 | Meenakshi | 62 | F | LE | Drooping | 129 | 211 | Mild NPDR | P | N | N | Full | 7 |
| 22 | Jayalakshmi | 52 | F | RE | Drooping | 165 | 254 | NAD | P | N | N | Full | 8 |
| 23 | Arunachalam | 65 | M | LE | Diplopia | 145 | 206 | Mild NPDR | N | P | N | Full | 7.8 |
| 24 | Lakshmi | 23 | F | RE | Diplopia | 68 | 110 | NAD | N | P | N | Full | 6 |
| 25 | Gopinath | 57 | M | LE | Diplopia | 154 | 222 | HTR | N | P | N | Full | 8 |

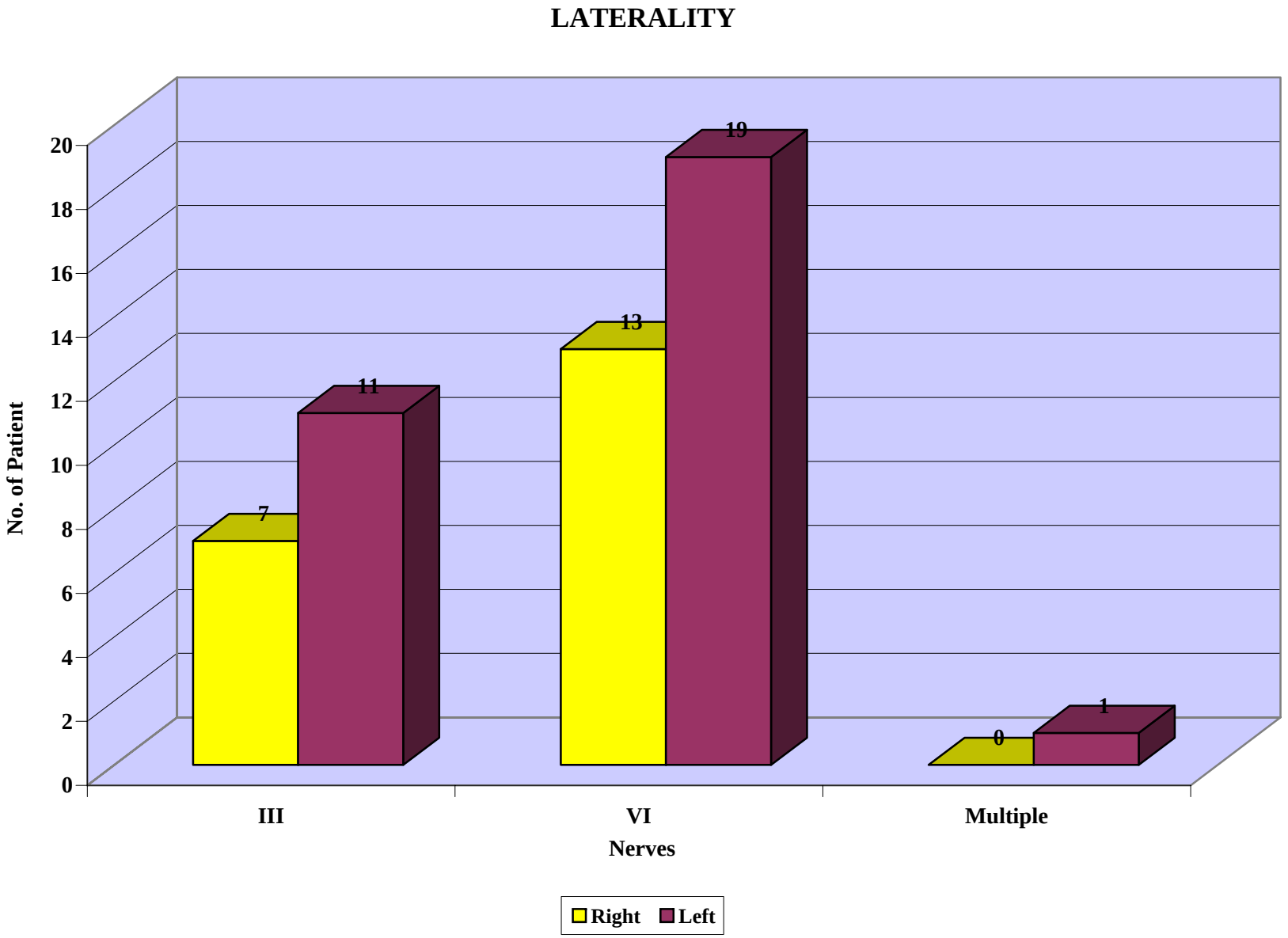
| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 26 | Rupalingam | 56 | M | RE | Drooping | 118 | 162 | Mild NPDR | P | N | N | Full | 8.5 |
| 27 | Rajammal | 60 | F | LE | Drooping | 200 | 296 | NAD | P | N | N | Lost | 9 |
| 28 | Saraswathi | 54 | F | LE | Drooping | 123 | 173 | HTR | P | N | N | Full | 7 |
| 29 | Krishnachari | 50 | M | LE | Diplopia | 177 | 256 | NAD | N | P | N | Full | 7.5 |
| 30 | Kathamuthu | 58 | M | RE | Drooping | 188 | 303 | NAD | P | N | N | Full | 8 |
| 31 | Gopalakrishnan | 66 | M | LE | Drooping | 218 | 342 | NAD | P | N | N | No recovery | 9 |
| 32 | Meena | 38 | F | RE | Drooping | 175 | 282 | Mild NPDR | P | N | N | Full | 9.5 |
| 33 | Muthusamy | 55 | M | LE | Diplopia | 144 | 228 | NAD | N | P | N | Full | 8.4 |
| 34 | Vijaya | 58 | F | LE | Diplopia | 118 | 192 | NAD | N | P | P | Full | 6.5 |
| 35 | Kala | 38 | F | RE | Diplopia | 106 | 168 | NAD | N | P | N | Full | 6 |
| 36 | Chokalingam | 56 | M | LE | Drooping | 212 | 200 | NAD | P | N | N | Partial | 8.5 |
| 37 | Gopinath | 35 | M | RE | Diplopia | 162 | 241 | NAD | N | P | N | No recovery | 7.5 |
| 38 | Elumalai | 70 | M | LE | Drooping | 149 | 193 | NAD | P | N | N | Lost | 6.5 |
| 39 | Ananad | 55 | M | LE | Diplopia | 176 | 242 | Mild NPDR | N | P | N | Full | 7.5 |
| 40 | Balamani | 57 | F | RE | Diplopia | 128 | 175 | ModNPDR | N | P | N | Partial | 8.5 |
| 41 | Thangarajan | 60 | M | RE | Diplopia | 147 | 204 | Mild NPDR | N | P | N | Partial | 9 |
| 42 | Nagammal | 39 | F | RE | Diplopia | 120 | 189 | NAD | N | P | N | Full | 7 |
| 43 | Panchatcharam | 65 | M | LE | Diplopia | 228 | 375 | NAD | N | P | N | Partial | 10 |
| 44 | Kamala | 57 | F | LE | Drooping | 147 | 290 | Mild NPDR | P | N | N | Partial | 7.5 |
| 45 | Rajasekar | 54 | M | LE | Diplopia | 125 | 249 | NAD | N | P | N | Lost | 8.5 |
| 46 | Sokkammal | 58 | F | RE | Diplopia | 78 | 118 | ModNPDR | N | P | N | Partial | 7.5 |
| 47 | Malakondaiah | 60 | M | LE | Drooping | 101 | 187 | Noview | P | N | N | Full | 6.5 |
| 48 | Vanaja | 45 | F | LE | Diplopia | 197 | 286 | NAD | N | P | N | Full | 7 |
| 49 | Nagapoornam | 55 | M | LE | Diplopia | 129 | 170 | NAD | N | P | N | Full | 8 |
| 50 | Mouiden | 55 | M | RE | Diplopia | 112 | 137 | ModNPDR | N | p | P | Partial | 6 |
| 51 | Ibrahim | 50 | M | LE | Diplopia | 206 | 311 | NAD | N | P | N | Full | 9.5 |

## AGE DISTRIBUTION

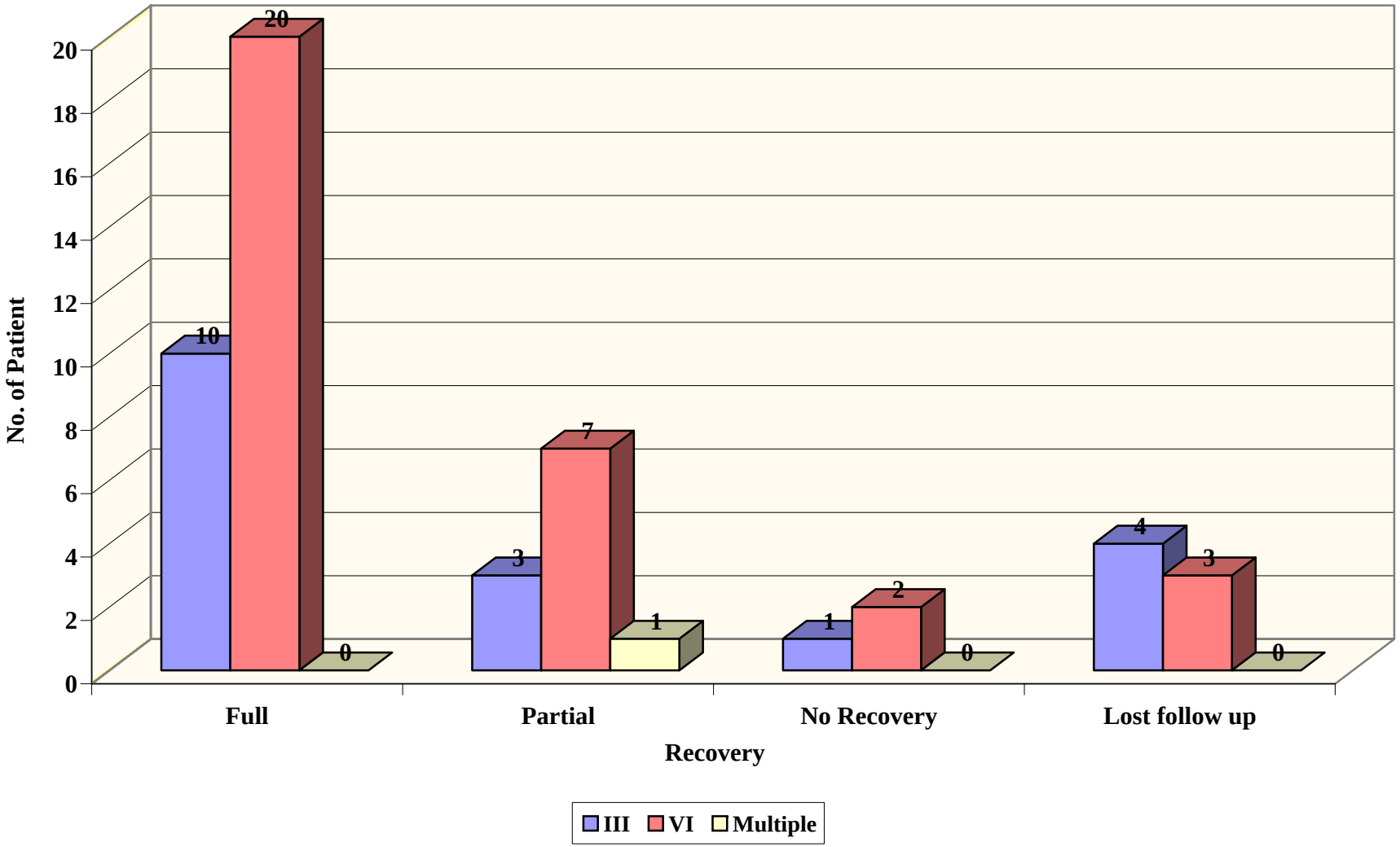


## SEX DISTRIBUTION

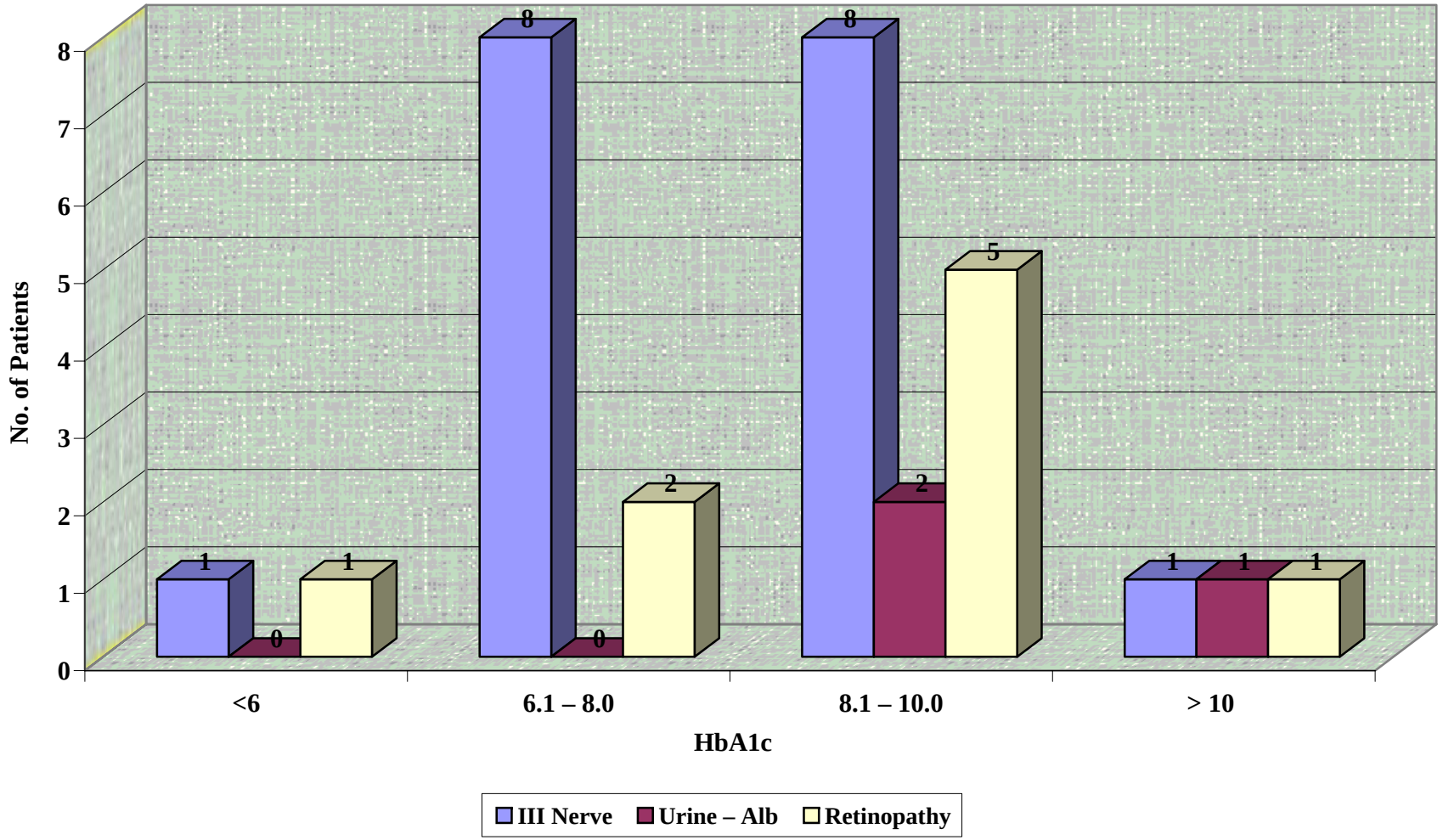



LATERALITY
No. of Patient
0
2
4
6
8
10
12
14
16
18
20
7
11
13
19
0
1
III
VI
Multiple
Nerves
Right
Left

## RECOVERY PATTERN



## GLYCAEMIC CONTROL AND MICROVASCULAR COMPLICATIONS IN III NERVE PALSY



## GLYCAEMIC CONTROL AND MICRO VASCULAR COMPLICATIONS IN VI NERVE PALSY